successful in bringing about considerable weight reduction and also resolution of symptoms and signs of IIH including reduction of CSF pressure to normal.

How does obesity lead to an increase in intracranial pressure? Hormonal mechanisms have been postulated,[70] but Sugarman *et al*[62] have provided persuasive evidence in morbidly obese people for a simpler mechanism. They showed that their obese patients with IIH had raised intra-abdominal pressures, raised intrathoracic pressures, and raised central venous pressures, supporting a direct cause and effect relation. However, they did not have a satisfactory, concurrent control group, and did not provide an explanation as to why the pulmonary artery pressures were higher in their obese patients with IIH than in equivalently obese patients without IIH. It would be easy to diagnose this patient group as just having a pickwickian syndrome, but only two out of eight morbidly obese patients of Sugarman *et al*[60] had evidence of alveolar hypoventilation with reduced blood oxygen and raised blood carbon dioxide concentrations. Obesity is common, whereas IIH is rare, and obesity affects males as well as females, whereas IIH is much commoner in females than males, so obesity cannot be a sole cause of IIH, even though it may be the immediate cause in a suitably predisposed person. The established relation between obesity and IIH raises further questions. Although IIH in obese persons may be rare, headache in people of all shapes and sizes is very common, and the question does arise as to whether a proportion of obese persons with headache but without papilloedema in fact do have IIH. In the classic IIH series, papilloedema was present in 100% cases and was a diagnostic sine qua non.[71–74] However, it is not necessary to modify the Dandy criteria much further to open up a whole new vista of cases of overweight patients with bad headache and with high CSF pressures. Indeed, only 12 out of 24 patients had papilloedema in the updated series of Sugarman *et al*.[63] Several centres have published on IIH without papilloedema.[75–77] Disappointingly, the response of the patients to medication aimed at lowering intracranial pressure was poor.

Conclusion

It seems inescapable that the condition currently called IIH is heterogeneous, and indeed Johnston *et al*[78] proposed using the term pseudotumour syndrome to encompass this heterogeneity. In some patients there may be just one aetiology operating, such as occult venous sinus thrombosis. Perhaps others have risk factors which combine to precipitate the condition. In particular, in many cases obesity may be a risk factor whereas in extreme cases it may be a sufficient cause. Brain MRI really should be able to provide definitive information about cerebral and CSF volumes in IIH, but as yet the tunnel has not shed much light, and intriguing enigmas remain. Laboratory animal research, if possible, into factors influencing function of the arachnoid villi might well be informative. More effective means of preventing or treating obesity would undoubtedly have an impact on the prevention and treatment of IIH.

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EDITORIAL COMMENTARIES

The volumes of memory

In 1937, Papez described a circuit for the processing of emotions, which has subsequently proved to be critical for memory function.[1] Various pathological entities can affect structures in this circuit, resulting in amnestic syndromes. In this issue of the *Journal* (pp 13–28), two related papers by Colchester *et al*[2] and Kopelman *et al*[3] used volumetric MR to assess the differing patterns of atrophy in patients with amnesia caused by several neurological diseases, and to examine the relation of these MR volumes to cognitive performance.

A great deal of recent work has focused on detecting atrophy in patients with neurodegenerative dementias, in particular Alzheimer's disease. Recent papers have reported significant atrophy in structures within the medial temporal lobe in memory impaired subjects even before the clinical diagnosis of Alzheimer's disease.[4–6] Relatively few studies have examined volumes of medial temporal lobe and other memory subserving structures in non-degenerative amnestic syndromes.

The report of Colchester *et al*[2] suggests that atrophy among the components of the circuit of Papez can be reliably quantified using MR volumetric assessment, and that amnestic syndromes of varying aetiology show specific patterns of atrophy. Of particular interest was the consistent finding of thalamic atrophy in the patients with Korsakoff's syndrome.

Complementing this work, Kopelman *et al*[3] examined the correlations between MR volumes of multiple brain regions and performance on several cognitive tests in patients with amnestic and other cognitive syndromes. Of note, the strongest relations were seen between hippocampal volume and anterograde memory measures, particularly evident in a factor analysis of the neuropsychological tests, although the thalamic measures did correlate with several memory tests.

These studies provide additional evidence for the critical role of the hippocampus in memory, and support the contribution of additional structures within the circuit of Papez to memory function. This work suggests that volumetric MR may be useful in elucidating the pattern of injury to neuroanatomical networks in various cognitive impairment syndromes.

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EDITORIAL COMMENTARIES

Long term effects of locomotor training in spinal humans

A major concern for patients after spinal cord injury is whether or not they will ever walk again. For completely paralysed patients this prospect is slim, but the outlook for those with partial cord lesions is more hopeful. Any programme, such as that suggested in the paper by Wirz *et al* (this issue, pp 93–96), which may increase the likelihood of subsequent ambulation is to be welcomed.[1]

Control of locomotion in primates is predominantly supraspinal. In the thalamic macaque monkey electrical stimulation of the posterior subthalamic region or the midbrain tegmentum just ventral to the inferior colliculi produced stepping movements.[2] This was abolished in the suspended animal if the cord was completely transected, even when reflexes had returned. However, if pathways in the ventral white matter on one or both sides of the cord were spared, stepping and walking did occur.

Locomotor training of patients with unilateral lower limb paralysis supported over a moving treadmill was found to improve power in the paralysed limb to make walking possible with minimal splintage and this was attributed to enhancing spinal interneuronal locomotor networks.[3] However, as the ventral part of the cord was intact, at least in part, this improvement could equally well arise from the training of higher centres, if not also to continuing recovery in spinal neurons, although unlikely in those patients who had started training many years after injury.

When this locomotor training was applied to patients with complete spinal cord injury, only four out of 10 showed an EMG response.[4] The failure of the remainder to respond was attributed to the drugs they had been taking—the adrenoceptor antagonist prazosin or cannabis. Support for this was obtained by giving intrathecal adrenaline (epinephrine) which increased and clonidine which decreased motor performance. It could also be possible that those who responded may have had some tracts functioning that could not otherwise be detected by clinical or electrophysiological techniques, but nevertheless allowed conduction to supraspinal centres.

The present study shows that in the partially paralysed patients there was an improvement in the amplitude of gastrocnemius EMG on completion of the treadmill training which was still maintained a mean of 2 years later, especially as all these patients continued to walk during this time. The completely paralysed patients could not walk so the smaller improvement in gastrocnemius EMG was lost when they no longer received treadmill training. The improvement during training never reached functional levels in these patients and advocating such intensive treatment without showing a functional benefit could be psychologically harmful. There may be some benefit if these patients were to proceed to a functional electrical stimulation walking programme after discharge from the treadmill training.

As has been advocated, it is important to compare novel interventions in treatment with conventional techniques.[5] Many treadmill studies lack adequate spinal cord injury control groups to demonstrate the value of such training and although spinal interneuronal responses may be stimulated, the vital contribution of higher centres cannot be ignored. With some current research focused on spinal neuronal regeneration, such as by the transplant of oligodendroglia, olfactory, or Schwann cells, any technique to increase the functional capacity of the receiving circuits must surely be encouraged.

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Cognitive function in the oldest old: women perform better than men

In the paper by van Exel *et al* in this issue (pp 29–32),[1] the authors examine the influence of sex and formal education on cognitive functioning in a community based sample of subjects over the age of 85. Based on the cognitive reserve theory of dementia, the authors hypothesise that women would be expected to score more poorly than men on cognitive tests due to a lower level of formal education.

Previous studies provide support for the theory that a lower educational level is a risk factor for the development of dementia.[2] This relation seems to be more pronounced

in female subjects than males,[3] although data regarding the influence of sex on cognitive functioning in non-demented persons were not available.

The results of the current study showed better cognitive performance in the female group, despite their lower level of formal education. One possible explanation raised by the authors was that medical risk factors (for example, atherosclerosis) may be greater in the male group. An alternative, or contributing factor, may be use of formal education as a measure of "cognitive reserve". Although years of education have traditionally been used to estimate premorbid functioning, some authors have suggested that formal education may be less important than later life experiences, such as primary occupation.[4] A follow up to the current study might examine the role of non-educational experiences on cognitive functioning between men and women.

This paper makes an important and timely contribution to the field of aging research, with the recent emphasis on early diagnosis of dementia. Understanding variables related to cognitive functioning in elderly people, such as the influence of sex, is essential for improving the ability to detect preclinical markers of dementia and identify "at risk" people who could benefit from clinical prevention trials.

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